June 7, 1950.

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Dear Roger:

Thanks for your inquiries re housing. I hope the Housing Office's optimisms warranted.

The maltose-glucose relationships in P. putrafaciens sound something like the Lac_1^- mutant story in E. coli K-12. This mutant is unable to adapt to lactose, but will adapt to butyl galactoside. If the effect is one of permeability, you have to postulate not only that the Glu+ mutant has now become permeable to glucose, but also that the preadaptation on maltose involves a similar change in permeability. I think that it will not take to strenuous a generalization to incorporate the permeability hypothesis into one which states our ignorance a little more clearly, namely the Stanierian. If there is going to be a difference in permeation of glucose and of maltose, then there must be a specific enzymatic mechanism of penetration. This is hardly distinguishable from the more general notion, of specific adaptation receptors. Some sort of test may be possible, however. If only permeability is involved, it should be possible to maintain and increase glucokinase in cells started on maltose, under the influence of glucose, under the same conditions as will the Glu+. This is less likely to be the case on a non-permability basis, but not impossible. These possibilities have always given me nightmares, but Occam's razor may be the only defensive weapon needed.

Have you thought of the parallelism between putrafaciens and E. coli W-327 re maltose? The latter cannot be adapted to ferment free glucose, but somehow the intact cells do manage to use all of maltase, if adapted, although the amylomaltase mechanism, and the dried cells, accumulate one mole of glucose. Have you been able to show directly in P. p. that glucokinase is formed in the Glu+ and not Glu- ? Has anyone followed up this story in W-327?

Sincerely,

Joshua Lederberg

P.S. Congratulations on the Lilly award.